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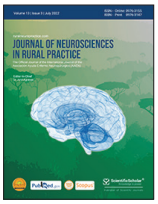

Case report

Improvement of gaze palsy and dizziness caused by vertebrobasilar dolichoectasia by conservative approach: A case report

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ABSTRACT

Vertebrobasilar dolichoectasia (VBD) is a rare case with incidence <0.05–0.06%. With prevalence ranges from 0.2% to 4.4%, its etiology is still unclear. We reported a case of a 62-year-old male brought to the hospital with complaints of sudden dizziness in the past 4 days before admission. Complaints were accompanied by binocular diplopia and a thick sensation on the tongue. There was no significant history of previous illness. The patient was prescribed dual antiplatelet therapy (100 mg of aspirin daily and 75 mg of clopidogrel daily) for 90 days, followed by continued single-antiplatelet therapy (100 mg of aspirin daily). After 5 days of treatment, the patient was discharged due to improved dizziness complaints. This case shows a conservative approach, blood pressure management, and stroke prevention, which could be considered as an initial treatment for VBD.

Keywords: Antiplatelet, Dolichoectasia, Gaze palsy, Vertebrobasilar

INTRODUCTION

Vertebrobasilar dolichoectasia (VBD) is a rare condition with an incidence <0.05–0.06%. With prevalence ranging from 0.2% to 4.4%, the etiology of VBD remains uncertain. Risk factors of VBD include older age, male sex, hypertension, dyslipidemia, atherosclerosis, smoking, and a history of myocardial infarction.[1-7] The clinical manifestations of VBD are diverse, ranging from cranial nerve involvement symptoms [e.g., hemifacial spasm (HFS), tinnitus, trigeminal neuralgia, ophthalmoplegia, and nystagmus] to headache, hemiparesis, parenthesis, dementia, ataxia, and even seizure.[1-4] Surgical intervention of microvascular decompression has been identified as a potential treatment to relieve the symptoms. Recent studies have demonstrated improvements in patient's symptoms. The following case report adds more evidence of an improvement in a VBD case with clear symptoms of dizziness accompanied by gaze palsy in VBD, managed noninvasively.

CASE REPORT

Male, 62-year-old, was brought to the hospital with complaints of sudden dizziness [dizziness assessment rating scale (DARS) = 6; Amer Dizziness Diagnostic Scale = 7] in the past 4 days before admission. Complaints were accompanied by binocular diplopia and a thick sensation on the tongue. There was no significant history of previous illness. The patient was an active smoker for approximately 10 years with a consumption of 1 pack/day.

Neurological examination revealed 3rd and 4th cranial nerve palsy of the left eye and right lingual palsy upper motor neuron type [Figure 1]. Basic laboratory results were only significant for hypokalemia (3.1 mmol/L). Head computed tomography (CT) scan imaging examination without contrast found arteriosclerosis with dolichoectasia of the basilar artery [Figure 2]. This finding was confirmed by cerebral angiography, which showed vertebrobasilar artery dolichoectasia, moderate stenosis in the proximal callosomarginal artery and distal left A2, and mild stenosis in the proximal segment of left A3 [Figures 3 and 4].

The patient was prescribed dual antiplatelet therapy (100 mg of aspirin daily and 75 mg of clopidogrel daily) for 90 days, followed by continued single-antiplatelet therapy (100 mg of aspirin daily). No interventional management was performed regarding dolichoectasia or stenosis in the patient. After 5 days of treatment, the patient was discharged due to improved dizziness complaints. Follow-up 5 months later, the patient's complaints of spinning dizziness and double vision were no longer present (DARS = 1). There were no obstacles to eyeball movement [Figure 5].

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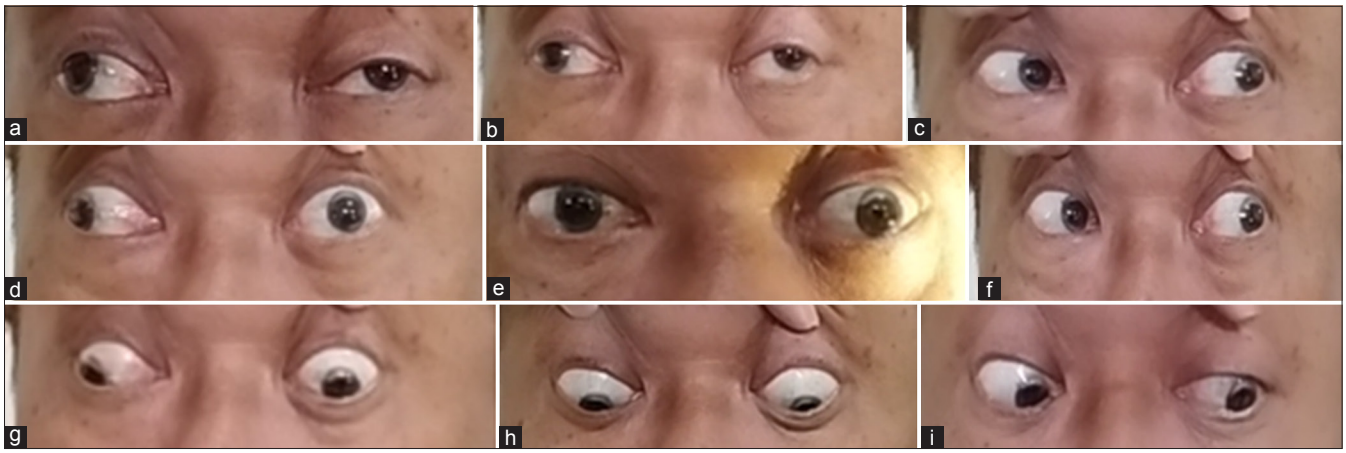

Figure 1: Ophthalmoplegia with involvement of left cranial nerve IV and partial cranial nerve III. (a-d,g) Left eye unable to gaze upward, medial, and medio-downward. (e) Frontal gaze showed the left eye slightly pulled to the lateral due to partial paralysis of the left cranial nerve III. (f) Patient still had normal left gaze. (h,i) Still able to gaze downward and latero-downward.

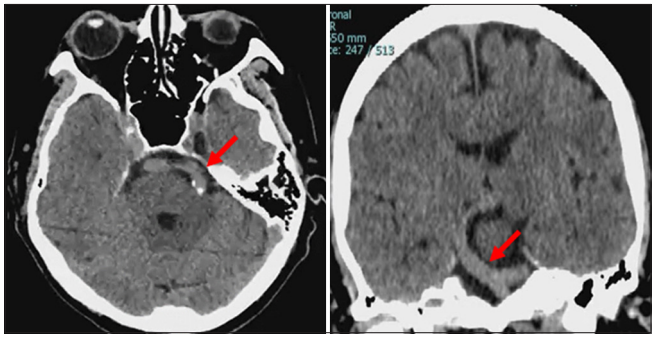

Figure 2: Non-contrast head computed tomography scan showed atherosclerosis with dolichoectasia of the basilar artery (red arrows).

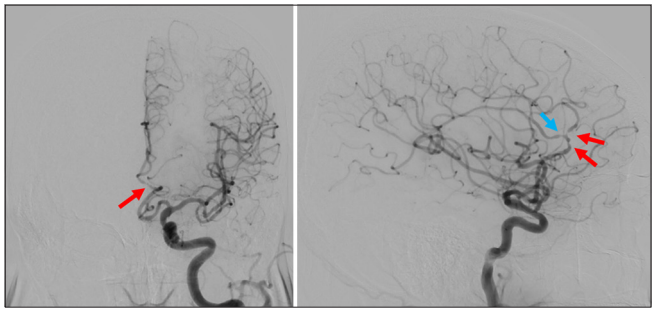

Figure 3: Cerebral angiography: Moderate stenosis in proximal callosomarginal artery and distal left A2 (red) and mild stenosis in the proximal segment of left A3 (blue).

DISCUSSION

VBD is a term derived from the Greek language, where "dolichos" means "elongation" and "ectasia" means "dilatation." It is a rare progressive arteriopathy primarily affecting intracranial posterior circulation, specifically the vertebrobasilar artery. It is important to differentiate between VBD and basilar dolichoectasia as they can present differently, different radiological features, and potentially

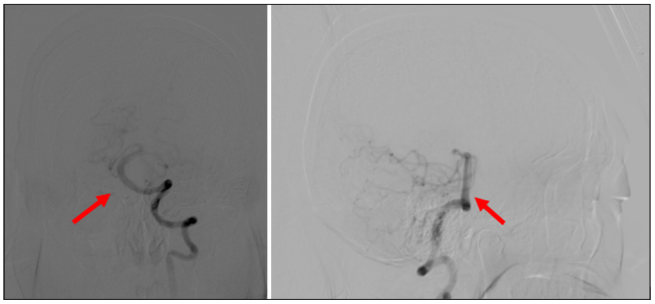

Figure 4: Cerebral angiography: Vertebrobasilar artery dolichoectasia (arrows).

result in different complications. The diagnosis of VBD is made radiologically with a basilar artery diameter at the midpons measured >4.5 mm, indicating dolichoectasia.[1] The diameter cutoffs for the vertebral artery ≥4 mm to denote ectasia. With prevalence ranging from 0.2% to 4.4%, the etiology of VBD remains uncertain. Old age, the male sex, hypertension, dyslipidemia, atherosclerosis, smoking, and a history of myocardial infarction have been proposed to be risk factors for VBD.[1-7]

The clinical manifestation of VBD can vary widely, from cranial nerve involvement symptoms (e.g., HFS, tinnitus, trigeminal neuralgia, ophthalmoplegia, and nystagmus) to headache, hemiparesis, paresis, parenthesis, dementia, ataxia, and even seizure. The underlying vascular mechanism leading to alteration in blood flow within the vertebrobasilar system results in decreased cerebral perfusion of posterior circulation territory due to limited space within the posterior fossa.[3] The trigeminal and facial nerves are particularly susceptible to involvement. Abducens nerve palsy resulting from VBD-related compression is rare, and multiple cranial nerve involvement in VBD cases is infrequently reported. For example, Madhugiri *et al.* described a case where the

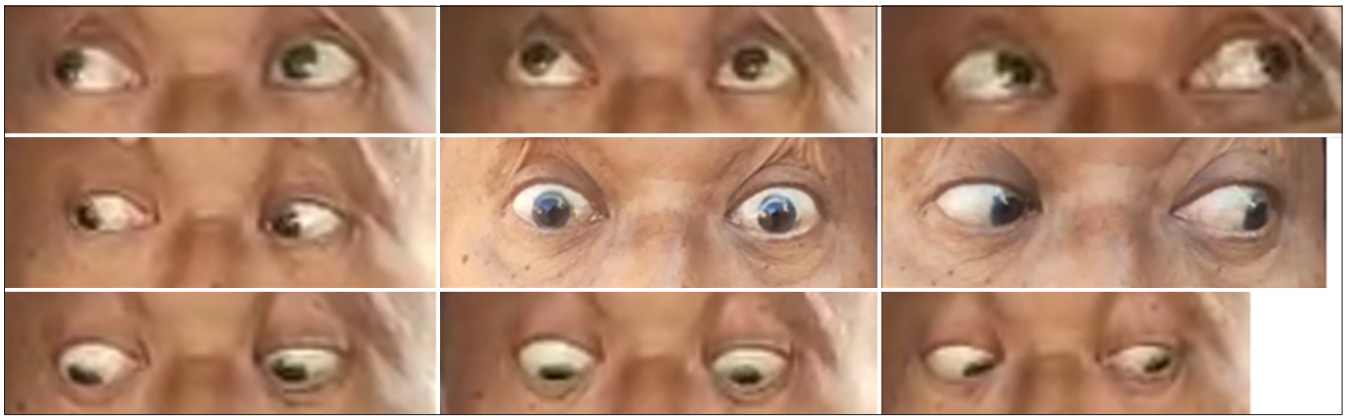

Figure 5: Improvement of left ophthalmoplegia after 5 months.

patient experienced diplopia due to a left abduction deficit, along with ipsilateral facial pain and muscle twitches.[6] These symptoms were attributed to compression of the abducens, trigeminal, and facial nerves by VBD. Pham *et al*. reported a similar case, where VBD causes both abducens and ipsilateral trigeminal nerve palsies.[7]

The diagnostic criteria for VBD have not been standardized and usually can be diagnosed incidentally during CT scans, cerebral angiography, or even magnetic resonance imaging (MRI).[2,8] Historically, autopsy findings were the main source of information on this condition. However, advancements in multimodal diagnostic imaging (such as CT/CT angiography, MRI/magnetic resonance angiography, and catheter-based angiography) have greatly improved our ability to diagnose VBD in living patients. The pathophysiology is typically attributed to the compression or indentation of nerve roots as they traverse the basal cisterns near their entry zone.

The mortality rate associated with VBD is 63% with a range of 1 month to 17 years. Common complications include ischemic stroke (17.6%), brain stem compression (10.3%), and transient ischemic attack (10.1%).[9,10] In some cases, surgical intervention involving microvascular decompression can be a solution to relieve the symptoms, though the evidence base remains constrained.[5] However, most reported cases have been managed conservatively with an emphasis on blood pressure control due to perceived risks associated with surgery. Supit *et al.* have documented the efficacy of blood pressure management in patients with clinical symptoms of HFS with VBD.[5] Furthermore, Sari *et al*. reported a significant improvement in stroke patients with VBD treated with IV thrombolysis. These results were also backed up with previous studies that reported no recurrence of ischemic stroke in VBD patients treated with antiplatelet.[4] Noted a pursuit in the controlled vascular risk factors must be optimized, as the use of antiplatelets or anticoagulants was associated with a 3 times greater chance of hemorrhagic stroke.[3]

CONCLUSION

Patients with dizziness and gaze palsy symptoms on VBD should be considered for neurovascular contact on the cranial nerve. The diagnosis can be incidental when the head CT is performed and should be confirmed by cerebral angiography or even MRI. However, an initial conservative approach is recommended before a surgical approach with comparable outcomes. Blood pressure management and antiplatelets or anticoagulants are the cornerstone in managing patients with VBD. Moreover, a high-resolution head MRI is useful to identify the neurovascular contact.

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